Thermal & pain

THERMOCEPTIVE SENSATION

Thermal Sensation:

Is the conscious perception of different grades of environmental temperature.

Types of Thermoreceptors:

1. **Warm**: Free nerve endings (**Ruffini** nerve endings) attached to **C** fibers.
2. **Cold:** Free nerve endings (**Krause's end bulbs**) attached to **C** & **Aδ** fibers.

In addition, two other types are stimulated by extremes of temperature:

3. **Cold pain**
4. **Warm pain**

Distribution of thermal receptors

1. Thermal receptors are located immediately under the skin, thus they respond to the temperature of the **subcutaneous tissue** surrounding them and not to the environmental temperature as such.

2. They differ in their distribution in different parts of the body: greatest in the lips, moderate in the finger tips and least in the trunk.

3. Cold receptors are **4-10 times** more numerous than warm receptors in any given area of skin.

4. They are **widely separated**. So, to differentiate between different degrees of temperature, a wide area of skin has to be exposed to allow spatial summation to occur.

Adaptation of thermal receptors:

Moderately adapting but warm receptors **adapt faster** than cold receptors.

- They respond markedly to changing temperatures rather than steady states of temperature:
- Warm receptors increase their firing when temperature is increasing.
- Cold receptors increase their firing when temperature is decreasing.

Detection of thermal sensation:

1- The human being can perceive different gradations of cold and heat, due to presence of 4 types of thermal receptors, ***(Fig.1):***

a- Cold pain receptors stimulated from 5-15 ^{o}C with maximum at 5 ^{o}C.

b- Cold receptors stimulated from 10-40 ^{o}C with maximum at 25 ^{o}C.

c- Warm receptors stimulated from 30-50 ^{o}C with maximum at 45 ^{o}C.

d- Warm pain receptors start to be stimulated at 45 ^{o}C.

2- At zero ^{o}C, no receptors discharge and a state of **anaesthesia** occurs.

3- **Comfort (or neutral) zone**: exists at skin temperature around 35^{-} ^{o}C where awareness of temperature disappears, due to equal discharge of both warm and cold receptors.

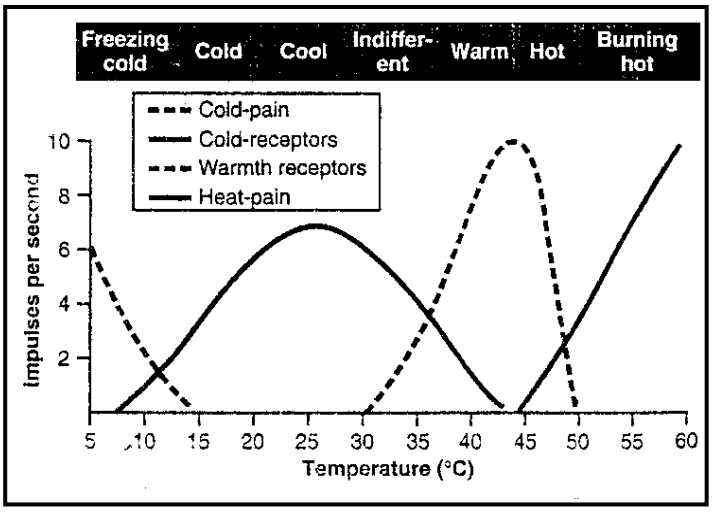

Fig. (1): Frequencies of discharge of:
1) a cold pain fiber 2) a cold fiber
3) a warmth fiber 4) a heat pain fiber

Thermo-receptive sensations

The pathway

The lateral spinothalamic tract.

PAIN SENSATION

Definition

Pain is an **unpleasant** sensory and emotional experience for **protection** of the body.

-It occurs whenever there is physical or potential **tissue damage.**

-It causes the person to react to remove the pain stimulus or seek medical advice.

PAIN RECEPTORS

Types of Pain Receptors: (Nociceptors)

Pain receptors are **Free Nerve Endings attached to Aδ and C fibers.**

They are classified according to the mode of stimulation into:

1- ***Mechanical pain receptors***: stimulated by mechanical injurious stimuli, e.g. cuts, bruises.
2- ***Thermal pain receptors:*** respond to extremes of temperature.
3- ***Chemical pain receptors:*** stimulated by chemical injurious elements *or* chemicals produced from tissue damage (**bradykinin**, **histamine**, **high acidity** & **environmental irritants**).
4- ***Polymodal pain receptors:*** respond to combinations of these stimuli.

Distribution of Pain receptors:

- They are most numerous in superficial layers of the skin.
- They are also numerous in peritoneum, pleura, periosteum, joints, arterial walls, dura and tentorium of the cranial cavity.
- They are less distributed in deep tissues and very few in Internal viscera.
- They are absent in liver parenchyma, lung alveoli, and brain tissue (***pain insensitive structures***).

Adaptation: slowly or nonadaptive receptors.

Pain Sensitizers (OR Chemical mediators of pain): (Fig. 2)

- After the nociceptors are stimulated**, the damaged tissues and the surrounding blood vessels** release a number of **pain** and **inflammation** producing chemical substances that are normally inside the cells, into the ECF.
- **These substances include** histamine, serotonin, K+, substance P, ATP, bradykinin and prostaglandins.
- These substances – in particular **Prostaglandins-** further **sensitize** the nociceptors, lowering their pain threshold, and producing the **primary hyperalgesia** that often accompanies pain.

- **Salicylates** and other non steroidal anti-inflammatory analgesics (NSAID) reduce pain by inhibiting prostaglandin synthesis.

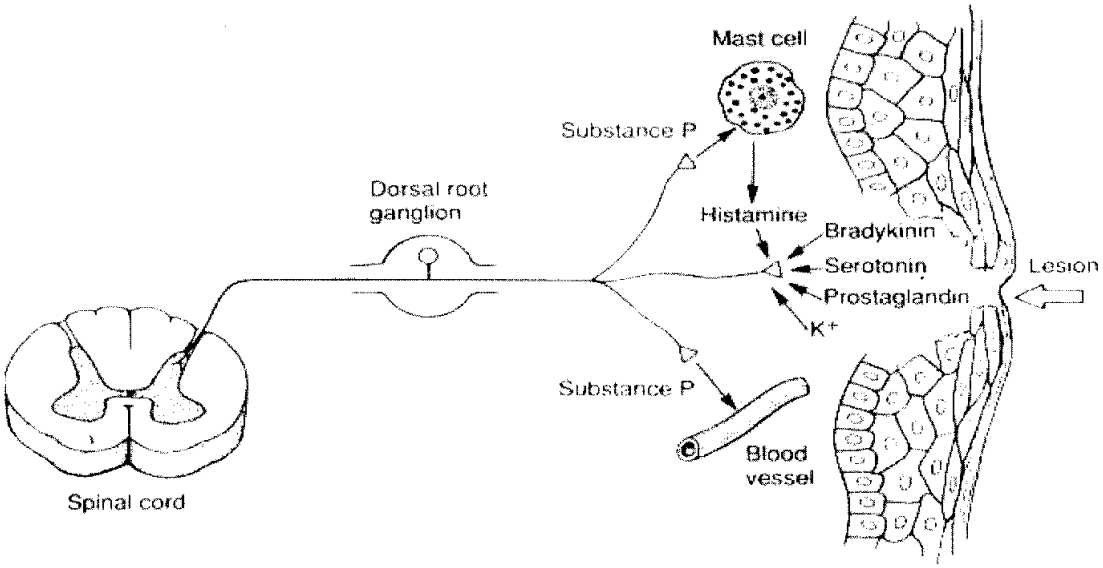

Fig. (2): Chemical mediators of pain

Cutaneous hyperalgesia

Definition:

It is an exaggerated response to a **noxious stimulus**. (increased pain sensitivity= an already painful stimulus now producing a more severe type of pain). **DD.** Allodynia ?

Types:

Point of view	Primary hyperalgesia	Secondary hyperalgesia
1-Site	In the **injured area**.	In the area of **healthy skin around** the injured area (not

		including the zone of injury)
2-The threshold of pain	Decreased	Normal may be even increased.
3-Duration of the lesion	Longer	Shorter
4-Mechanism	***Sensitization* theory** Local pain receptors are sensitized by the release of inflammatory mediators eg. prostaglandins.	***Convergence- facilitation theory*** -Afferents form both the health and the injured area converge on the same DHC. -Impulses from the damaged area facilitate the DHC which give more afferent discharge on arrival of the pain impulses from the healthy area.

Types of pain

/- ***According to site of origin of pain*:**

a) **Cutaneous pain** due stimulation of pain receptors in skin or body surface.

b) **Deep pain** due stimulation of pain receptors in deep structures as muscles, tendons and joints.

c) **Visceral pain** due stimulation of pain receptors in viscera.

//- ***According to quality of pain*:**

a) **Fast pain**: also called sharp, immediate (**Aδ fibers)**

b) **Slow pain:** also called dull aching, delayed. (**C fibers)**

	Fast Pain	**Slow pain**
Quality	Sharp, pricking, acute	Dull aching, Burning, throbbing

Onse	Immediate	Delayed
Duration	Short duration	Long duration and increase with time
Localization	Well localized	Poorly localized (diffuse)
Stimulated Receptor	Mechanical & thermal pain receptors	All types of pain receptors
Felt	Skin and Parietal surfaces	Skin, deep structuresnand viscera
Carried by	Aδ fibers	C fibers
Blocked by	Hypoxia and compression	Local anaesthesia (e.g. cocaine)
Neurotransmitt	Glutamate	Substance P
Pathway	Neo-spinothalamic Tract	Paleo-spinothalamic Tract
Relay of 2nd order neurons	PVN of thalamus	Reticular Formation → intralaminar thalamic N
Termination of fibers	sensory cortex	whole cortex
Pain	In Thalamus and Sensory Cortex	Mainly In Thalamus
Motor reflexes	Withdrawal reflex	Guarding: hypertonia of overlying muscles
Autonomic	Pressor response: (↑B.P. and H.R.)	Depressor response: (↓B.P. and H.R.) Nausea and vomiting
Emotional	Anxiety	Depression

A] <u>CUTANEOUS PAIN</u>:

- It is pain produced by stimulation of pain receptors in the **skin or body surface.**
- It starts as fast sharp pain followed by more prolonged slow dull pain.
- Unlike the other types of pain, <u>it is accurately localized, due to:</u>
  a) The high density of pain receptors in the skin.
  b) The fast pain fibers reach the sensory cortex.

c) Besides, touch and vision help greatly in localization.

B] <u>DEEP PAIN</u>

Deep or musculoskeletal pain is the pain produced from injury to muscles, tendons, ligaments, joints and bones. It is conducted along thin **C fibers**.

<u>Causes:</u>

(1)<u>Trauma</u>: e.g. broken bone.

(2)<u>Inflammation</u> in deep tissues or joints: e.g rheumatic arthritis.

(3) <u>Muscle spasm</u>: e.g. injury to bones, tendons and joints is associated with reflex contraction of nearby skeletal muscle → ischemia→ more pain → spasm → ischemia→ pain …etc (vicious circle).

(4) <u>Ischemia</u> block of blood flow to a tissue leads to ***<u>Ischemic Pain</u>***:

a- Ischemia may be caused by narrowing or compression to an artery by thrombosis, spasm or mechanical pressure by a tumor.

b- Pain is produced by **accumulation of metabolites and proteolytic enzymes in ischemic tissue.**

b- Pain is aggravated by increased metabolic rate of the affected organ and relieved by rest.

c- Example: Anginal pain in cardiac muscle and Intermittent claudication in skeletal muscles.

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